

## NATURAL VERSUS ANTHROPOGENIC DISTURBANCES TO CORAL REEFS: COMPARISON IN EPIDEMIOLOGICAL PATTERNS OF CIGUATERA.

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Patterns of ciguatera fish poisoning vary from one location to another. In French Polynesia surveys have shown that outbreaks of the disease are associated with disturbances to live coral reefs. Anthropogenic damages such as undersea works, dumping of wastes, wreckage of ships, crashing of ship anchors may result in a flare up of poisonings in areas with no previous history of ciguatera; in this pattern, detritus-feeding herbivorous fish or invertebrates and carnivorous fish may become toxic in a confined area over variable periods of time. Natural catastrophes such as hurricanes, tsunamis, massive coral bleaching may be associated with a pattern of diffuse continuous risk of ciguatera poisoning from large predaceous fish, with periodic outbreaks involving fish from primary and secondary trophic levels. Seasonal disturbances such as storms, heavy swells, high freshwater drainage, red tides, seem to be consistent with a pattern of ciguatera poisoning in which the overall picture is stable, with the same fish species, most of the time large predators, toxic in well defined, extensive areas.

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In French Polynesia most islanders depend greatly on seafood; increases in toxicity of local fish does not go unnoticed. For decades, knowledge of ciguatera fish poisoning in most areas was based on information of variable reliability gathered from a diverse range of persons who had been in contact with the disease. These included medical staff treating it, persons afflicted with it, fishermen and administrators.

Fish poisoning was listed among the notifiable diseases to South Pacific Commission in 1974. Although the reporting is for all types of fish poisoning, it may be assumed that most cases deal with ciguatera (the pathognomonic cold-to-hot sensory reversal dysthesia makes it easier to distinguish from other types of fish poisoning). Reports from French Polynesia are completed by the Public Health Department staff (PHD) in Papeete and mailed, each month, to the South Pacific Epidemiological and Health Information Service (SPEHIS). Reporting is compulsory for each territorial Health Department Unit and voluntary for private medical practitioners. In both cases, it relies on the person in charge to provide accurate and timely monthly reports. Delays in reporting cases are beyond the control of both PHD and SPEHIS. Due to forwarding and transmission of mail, an outbreak occurring today may be notified one or several months later. So seasonal evaluation from rough official data is

questionable. The number of cases reported depends on several factors. Severe cases that occur near a health unit are most accurately reported, whereas mild poisoning on the various islands without medical or paramedical staff is probably less accurate. Cases treated according to traditional medicine may also not be notified. Moreover, simplified official data do not provide information on the name or species of the accused fish, nor on the location of its capture.

Cases reported during 1974-1990 give an indication of the magnitude of the ciguatera problem in French Polynesia compared to other Pacific countries reporting in the same way. Data available at the PHD show that no island was completely immune from it, but morbidity is not evenly distributed and the overall data are not always significant for the region as a whole. For instance, data recorded in Tuamotu Islands are less accurate than those from the Gambier Islands. The 12,500 inhabitants of the Tuamotus live on 46 atolls separated by a few or by many miles of ocean and are spread over 400,000 square miles; they are served by two mobile medical practitioners, and have at their disposal one hospital in the main atoll of Rangiroa and five infirmaries located in Anaa, Fakarava, Hao, Makemo, Reao, notwithstanding the military medical staff in Hao and Moruroa. On the other hand, the Gambiers consist of 10 small volcanic

islands in one 600 square mile lagoon; the 600 inhabitants of the archipelago are grouped in one island, Mangareva, with an infirmary in the main village.

To assess accurately the qualitative, quantitative and time related aspects of evolution of ciguatera in the French Polynesian islands, a monitoring programme was developed from 1965 to 1990 by the author and the Malardé Institute staff.

### MATERIAL AND METHODS

From 1965, a daily follow-up of cases in the whole territory was set up using the medical and paramedical staff of the Public Health Units, with the filling in of 15,000 standard questionnaires consisting of 26 medical and epidemiological parameters.

From 1967, nearly 20 tons of fish, caught in 30 different French Polynesian Islands exposed to ciguateric risk, and belonging to 30 families from various trophic levels, were bioassayed for ciguatoxicity. From 1976, 10,000 grams of macro-algae, among 40 species, were sampled for research and counting of *Gambierdiscus toxicus* cells.

Follow up of these various analyses, over 15–25 years, resulted in 3 indexes to monitor ciguateric risk (Bagnis et al., 1985a): the number of cases per 1,000 residents (CIR), percentage of toxic individuals in a group of fish from a given species, family or trophic level (PCI), density of the populations of the toxic dinoflagellate *G. toxicus* per gram of algae (GTD).

In addition to these research programs, the author had many discussions on fish poisoning problems with officials from fisheries and health departments, old natives and longtime residents, to assess what events, according to them, could be associated with ciguatera.

### RESULTS AND DISCUSSION

Within the context of the monitoring programme, in each inhabited atoll or large valley, a person was officially appointed to fill in the questionnaires and, from 1965 to 1990, the ciguatera reporting system in French Polynesia has gradually become one of the most comprehensive in all of the Pacific area. It has been estimated that 70–80% of cases are reported (instead of 10–20% registered by the PHD in Papeete, from 1960 to 1965). During that time, about 30,000 cases of ciguatera were recorded

(CIR: 8 ‰), caused by some 100 fish species from various trophic levels. Gambier Islands (11% of cases; CIR: 60 ‰), Marquesas Islands (19% of cases; CIR: 30 ‰), and Tuamotu Islands (17% of cases; CIR: 25 ‰) were the most involved, compared to Society Islands (52% of cases; CIR: 5 ‰) and Austral Islands (<1% of cases; CIR ‰). As patterns of ciguatoxicity may vary in time and space, from one island to another and from place to place in the same island, only data provided by the questionnaires give an account of the situation in each separate island. Thus, in the Tuamotu Islands, the average overall CIR was influenced by several flare ups that occurred in Hao (primarily) but also in Hikueru, Moruroa, Takaroa, Takapoto, Manihi, Reao, Fakarava, Mataiva, Anaa, Makemo from 1964–1975. In the Gambier Islands, from 1971 to 1980, the CIR never decreased below 300, reaching 560 at its highest point in 1975. In the Marquesas Islands, the CIR grew from about 10 in 1963 to 80 in 1971, and started decreasing gradually in 1976. In the Society Islands an outbreak occurred from 1964–1970 in Bora Bora, while at the same period a significant increase of the ciguatera poisonings by fishes caught in the area of Faaa, was reported in Tahiti, with a high surge in 1966–1967. In the Austral Islands, the least affected of the Polynesian archipelagoes, a few cases are episodically reported from each island but Rapa. In French Polynesia, there are also islands where for decades the ciguateric risk seems to be limited to some species of fish and to some well defined areas, with a level of ciguatoxicity apparently unchanged (Bagnis et al., 1985a).

The most significant data about the PCI concern Tahiti, Hao, Gambier and Marquesas Islands. In Tahiti, the study was carried out from 1967 (at the peak of the surge) to 1984, on the most frequently poisoned fish, the surgeon fish maito, *Ctenochaetus striatus*, and showed that the average PCI was cut by half during that period. In Hao, the PCI increased from c.0 (in July 1966) to c.70% (within 2 years) before decreasing from 1970 to reach 7.5% in 1982. In Gambier Islands, from 1969 to 1971, PCI increased five-fold to a maximum of 67% during 1975–1978, for detritus-feeding herbivorous fish and tenfold to a maximum of 80% during 1978–1979 for carnivorous fish, before decreasing gradually. The data related to Marquesas Islands indicate, from 1968–1984, a regular increase of PCI for herbivorous and carnivorous fish, from respectively 4.5 to 26% and 5.3 to 22% (Bagnis et al., 1985a).

A close relationship between PCI and GTD

could be observed in the Gambier Islands (Bagnis et al., 1985a) and on Hitiaa Reef in Tahiti (Bagnis et al., 1985b). In the Gambiers, where data were the most significant, the yearly average of GTD decreased gradually from 45,000 in 1977 (a year that fits roughly with the highest values of the PCI) to 40,000 in 1978, 10,000 in 1978–1980, 1,500 in 1982, <150 in 1984 to c.100 in 1989–1990. Elsewhere, the follow up was not accurate enough to allow comparative evaluation between the two indexes (PCI and GTD), within the same time span.

Previous data and several events associated with ciguatera, either from the monitoring of some flare-ups, or noted empirically by natives, such as various well identified anthropogenic and natural disturbances in the coral reef ecosystem (Bagnis, 1987), support the hypothesis that the appearances of poisonous fish are directly linked to changes in some factors of their environment (Randall, 1958). Helfrich & Banner (1968) suggested that the patterns of ciguatera in the Pacific fall into one of three main categories or occasionally a combination of these. These patterns are discussed below with reference to ciguatera in French Polynesia.

#### EPIDEMIC PATTERN

This pattern of poisoning concerns areas with no or a rare history of ciguatera. In such areas, after the initial outbreak, there was a general increase in toxicity reaching a peak of severity (based on the number of species affected and the level of the PCI) within 2–5 years. After some time, the toxicity began to decline. In the islands of French Polynesia where such flare-ups have been observed, this occurred c.5–10 years after the onset of toxicity. The decline may start sooner, but its exact timing is often obscured by i) the fact that the local population is reluctant to begin eating a fish after most of its members were stricken in the period following the initial outbreak (several months or years) and ii) by frequent clinical hypersensitization features which can be mistaken for true poisonings. The rate of decline in ciguatoxicity varies also according to areas. Grazing or browsing herbivores and detritus feeders, such as some acanthurids (*C. striatus* or *C. strigosus*), scarids (*Scarus gibbus* or *Scarops rubroviolaceus*) or mugilids (*Crenimugil crenilabis*), become toxic first, followed within a few months by the carnivores at higher trophic levels (serranids, lethrinids, lutjanids, carangids, labrids, muraenids). Thus the various links of the food web are progressively

involved. At the peak of the flare up, an island that had not harboured any toxic fish in man's memory, may have all fish contaminated within a year, according to the natives. In the declining toxicity phase, herbivores become significantly less toxic first, followed by some species at higher trophic levels, until only a few of the large carnivores remain toxic.

Most flare-ups in the Tuamotu Islands, since investigations started, proceed with this pattern (Bagnis, 1969, 1982; Bagnis et al., 1973, 1985a). Similar features have been observed in Society Islands, Austral Islands and Gambier Islands (Bagnis et al., 1988).

All these flare ups followed various anthropogenic damage to coral reefs, linked to public or military works (Bagnis, 1987). The main disturbances were: tearing, blasting, disrupting, scouring of pieces of coral reef (to deepen or widen passes in the barrier reef, to open channels in lagoons, to drill shafts in the basaltic layer for nuclear tests), crashing of heavy ship anchors and ploughing caused by dragging anchor cables, dredging and shifting of sand, dumping of wastes or debris (chiefly metallic ones), wreckage of ship, building of piers, wharfs, roads, protective sea-walls on live coral reefs and any other damage less evident and more traditional, like massive diving for pearl-oysters, or usual surf-landing of whaleboats on the same outer reefs of atolls without a pass. Usually, human damages induce geographically limited flare ups (Bagnis, 1969, 1981; Bagnis et al., 1973). In the Pacific area, outbreaks of fish poisoning during the Second World War were very likely related to man-made damages on coral reef (Halstead, 1967).

#### ENDEMO-EPIDEMIC PATTERN

This pattern of poisoning does concern areas with a history of continuous ciguateric risk for the local population, made up of periods of stable, relatively low fish toxicity, referred to as the 'quiescent stages' by Cooper (1964), alternating with periods of major outbreaks, usually only within the memory of the older inhabitants.

In this pattern, toxicity remains confined, during long periods, to large carnivorous fish: some snappers, groupers, jacks, emperor fish and moray eels. Periodically, such areas experience flare ups similar in form and duration to that described for the Epidemic Pattern. Thus, the detritus feeding herbivores (like some surgeonfish, parrotfish, mullets) become toxic, followed by some carnivores at higher trophic levels,

which were previously safe. Finally, all the food chain may be more or less affected by ciguatoxicity during a period of 5–20 years in extensive parts of the coast (Bagnis, 1974). This pattern was first observed in Marquesas Islands where data obtained by the author (1965–1973) from inquiry with old natives, pointed out 4 flare ups since the beginning of the century. After multiple cross-checking, their dates and durations could be roughly established: 1905–1915, 1925–1935, 1953–1958, 1965–1985. The first broke out, according to the people interviewed, after the passage of some cyclones. Such an assumption prompted the author to look for a chronological relationship between the alleged flare ups and cataclysmic events, in these islands not protected by a barrier reef and occasionally exposed to tsunamis. Informations collected from the Geophysical Laboratory of Tahiti, the French Meteorological Service and the Hydrographic Department of the French Navy about tsunamis, strong storms, hurricanes which have affected Marquesas Islands since the beginning of the century, pointed out a close relationship between both sets of events (Bagnis, 1980). Some data from Majuro in the Southern Marshall Islands (Bartsch & McFarren, 1962), in certain Kiribati Islands (Cooper, 1964), could also illustrate this pattern.

The episodic resurgence of toxicity involving detritus-feeding herbivorous fish, would indicate that *G. toxicus* (or another ciguatoxic microorganism) was recently (or is still) proliferating again and actively manufacturing the ciguatoxin or a precursor of it, very likely amongst the widespread macro-algal turf covering the many coral colonies damaged by the passage of a tsunami or a cyclone. This new toxin production would increase the overall ciguatoxicity of the food chain for years.

The long quiescent periods, during which only a few large carnivores are toxic, may be explained by the longevity of the fish themselves and their ability to retain toxin. The available data about longevity of tropical reef fish, indicate that some may live more than 30 years (moray eels). If the ciguatoxin were restricted to a 'pool' in the ecosystem bound in the organisms at trophic levels above the secondary one, and the reduction of toxin in the pool would be only by means of natural mortality and break-down by reducer organisms, assimilation by other consumer organisms would merely retain and recirculate it within the pool. As the overall level of the ciguatoxin in the pool slowly declines through

loss of toxic individuals by natural mortality, reduction by microorganisms and possibly by slow natural excretion, only those animals with very great longevity would be expected to be toxic, and to contain a large quantity of ciguatoxin (Helfrich & Banner, 1968).

Another example of potential recycling of the ciguatoxin in the pool of carnivores in a restricted area is frequently observed in Marquesas Islands (and elsewhere). Many lutjanids, serranids and lethinids, considered as toxic in some areas, are thrown back into the water when caught. Such a practice would essentially act as a feedback mechanism in the ecosystem. In this condition, one can think that, rather than a gradual accumulation of the toxin at the higher trophic levels by the carnivores with the greatest longevity, as has been noted previously, the activity of the fishermen may continually recycle the toxin among the carnivores, favouring those with non-specialized food habits, such as the scavengers (some lethinids for instance).

Another explanation of a resurgence in the ciguatoxicity (in both the level and the species involved) of reef fish during the 'quiescent stages' lies also in some anthropogenic disturbances. Thus, extensive blasting and dredging may release some of the ciguatoxin concentrated in the pool of sedentary eels, groupers, wrasses and distribute it to individual usually safe carnivorous fishes at various trophic levels in the community. Such events may result in a series of scattered cases of ciguatera incriminating fish other than detritus-feeding herbivorous fishes.

#### ENDEMIC PATTERN

This pattern occurs in areas in which the overall picture of toxicity seems to be quite stable, with the same species (most of the time predators) exhibiting the same level of toxicity within well-defined geographical areas. The condition is said to be unchanged as far as the local people can remember. Confirmation of the continued toxicity of the fish usually occurs when an outsider, such as a tourist or a member of a ship's crew, is poisoned or when a native who cannot resist the temptation offered by a meal of a fat, succulent grouper, snapper or eel, gamble on it being nontoxic and lose. According to old islanders, approximately the same species (most of the time carnivorous, but also detritus-feeding herbivorous fish) that were toxic in some reefs of Society, Marquesas, Tuamotu, Austral and Gambier Islands 50 years ago, may still be toxic today. The same stability is observed outside of French

Polynesia in some parts of New Caledonia, Vanuatu, Fiji, Samoa, Tonga, Guam, Marshall Islands, (for the Pacific), Reunion, Mauritius (in Indian Ocean) and in most Caribbean Islands.

Seasonal natural disturbances like heavy swells, abundant rains with freshwater drainage and soil runoffs, red tides, slight coral bleaching, crown of thorns proliferation, and any other kind of insidious disturbance, not taken into consideration by man, could be the most frequent causes of this pattern of ciguatera poisoning, with recycling of the toxin by the fishermen. Every disturbance destroying coral colonies and creating consequently new surfaces available for macro-algal colonization results either in a significant increase of genetically toxic strains of *G. toxicus* or in the development of toxicity by previously non toxic strains, because of some changes, very likely due to the microflora associated with *G. toxicus*.

From today's knowledge on the origin of ciguatera, natural disturbances would explain its antiquity and its occurrence in many uninhabited islands and shoals distant from land. Through information provided by old islanders, one may safely say that no island of French Polynesia, save perhaps Rapa, is completely immune from it.

### CONCLUSIONS

To understand the dynamics of the evolutive patterns of ciguatera, one should remember that the reef community is probably the most complex in the sea. A delicate balance must exist among competing organisms, so much so that a subtle change in environment can result in temporary proliferation of one at the expense of the others. Whatever the disturbances in lagoon, pass, fringing or barrier reef, the result is the same: occurrence of dead coral beds and new surfaces available for opportunist populations. We know that toxic *G. toxicus* is very poor in reef, in normal environmental conditions; but it may increase rapidly and significantly among the filamentous or calcareous algae growing on new or denuded surfaces, in normal ecological succession, fixing itself on the thalli of the algae, primarily the red ones. If we postulate *G. toxicus* as the single or main source of ciguatoxin, with herbivore ingestion as the chief means of transfer of the toxin to higher trophic levels, then the more widespread the macroalgal colonization, the more the toxic dinoflagellate bloom can be important and the higher the amount of toxin introduced and stored in the food-web. The detritus-feeding her-

bivorous fishes can be expected to remain poisonous, after the ciguatoxin producing dinoflagellates decrease in number, disappear or cease to be toxic, by a period of time equal to the maximum longevity of the fish species involved.

Discussion on patterns of ciguatera development is based on empirical data, from French Polynesia. It is admittedly tenuous and conjectural in some cases. Nevertheless, it supports Randall's (1958) hypothesis, resumed by Helfrich & Banner (1968), on the evolution of ciguatera.

The epidemic pattern seems to be most of the time associated with anthropogenic damage (Bagnis, 1969, 1982; Bagnis et al., 1973). The endemo-epidemic pattern may be related to natural catastrophes, as in the Marquesas Islands with their fringing patches of coral without the protection of barrier reefs and many shoals away from the shore, especially exposed to variations of oceanic hydrodynamism (Bagnis, 1980). The endemic pattern may be associated with natural seasonal disturbances in most of volcanic islands with fringing or barrier reef where the detrimental action of man or cataclysms in the marine environment are negligible (as observed in many parts of French Polynesia). The combination of the three patterns is observed in Gambier, where all the islands are edged with a fringing reef and bounded with the same continuous half emerged 168 miles long barrier reef; the latter is bordering a relatively shallow lagoon, with a bottom rugged with many patch reefs, knoll reefs and coral ridges. In this archipelago, a close relationship between ciguateric fishes and anthropogenic and natural damage to reefs was pointed out (Bagnis et al., 1988).

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